the right ovary that was removed), accompanied by induration in the course of the femoral vessels from the groin to the popliteal space. The patient improved gradually, so as to have very good health during the last month of pregnancy. Dr. Galabin, of Guy's Hospital, who was the operator in this case, says :—

> "It is difficult to trace a direct causation for the phlegmasia dolens by which my patient was attacked fifteen days after the operation. If there had been any access of septic material, notwithstanding the carbolic spray, she would not have progressed so favourably for fourteen days after so severe an operation; and if the starting-point were inflammation near the pedicle, set up by the presence of the ligatures, it might have been expected that the right leg, and not the left, would have been affected."

Of course, in my case the presence of ligatures is to be left out of consideration altogether, in looking for the causes of the inflammation of the vein, and I am quite inclined to believe that the trouble did come from septic, or more properly, from *irritating* material, and see no reason whatever to doubt this from the fact that the cases did well for some ten days or two weeks. The irritating material was, I believe, the peculiar fluid in the sac, which in both cases had the same characters, and some of which, as before said in my case, got access to and remained in the abdominal cavity. Dr. Galabin says of this fluid that "it was not pale, but was stained by blood pigment to a deep reddish-brown," and "that the whole interior of the cyst was covered by papillary growth." The cysts and their contents had the same characters in these two cases, and I would attribute the extraordinary complication that attended them both, to the influence of the fluid that certainly escaped into the cavity of the abdomen in one case, and that may have done so in the other.

ARTICLE VII.

ON THE PHYSIOLOGICAL PATHOLOGY OF THE BLOOD.

By THOMAS WHARTON JONES, F.R.S., F.R.C.S., Professor of Ophthalmic Medicine and Surgery in University College, London; Ophthalmic Surgeon to the Hospital, etc.

IN a communication to the *Lancet*, two or three years ago, I showed that the process by which arrestment of bleeding from divided vessels in the bat's wing or frog's foot spontaneously takes place, as observed in detail under the microscope, is substantially similar to the process in the case of larger vessels of larger animals as explained by Dr. J. F. D. Jones,[1] in the beginning of this century. I pointed out, as Dr. Jones had done before, how cessation of bleeding is, in the first instance, owing to the mouths of the divided vessels becoming stopped up with clots of blood, and that this is favoured by the retracted and constricted condition which the mouths of those vessels themselves assume.

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[1] On the Process of Nature in Suppressing Hemorrhage from Divided and Punctured Arteries, London, 1805.

Coagulation of the blood, though in some respects a moribund manifestation, thus serves here an important preservative purpose, and may be appropriately accepted as constituting the first great point calling for consideration in a discussion of the physiological pathology of the blood.

The blood, as observed under the microscope, circulating within the vessels of the web of a bat's wing or frog's foot, for example, is seen to consist of the liquid plasma and the corpuscles—red and colourless—suspended in it.

The coagulation, which the blood undergoes after it has been drawn from the living body, is owing to a precipitation from the plasma of the stringy substance, named fibrin. This substance, according to the chemical doctrine at present prevailing, does not pre-exist as such dissolved in the plasma of living blood, but results from the combination of two different albuminous substances contained in solution in that fluid, namely *fibrinogen* and *fibrinoplastic.* The combination of these two substances to form solid fibrin, after the blood has been drawn from the living body, is supposed to be brought about by the agency of a ferment which is alleged to be derived from the contents of the colourless corpuscles of the blood set free by the bursting of those cells. Colourless corpuscles, however, do not really burst, though they collapse and undergo changes of shape by shooting out their wall in processes like the fingers of a glove. Their contents, therefore, are not evacuated.[1] And if a ferment, causing the combination of fibrinogen and fibrinoplastic to form fibrin, be generated in the blood, after being drawn from the body, it must be derived from some other source.

The fibres of the fibrin of coagulated blood are interwoven like a feltwork, in the interstices of which the corpuscles and serum are inclosed; but by and by the serum, which is the residue of the plasma, oozes out by the shrinking of the fibrin, whilst the corpusles are retained.

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[1] See the descriptions and delineations of this in my Papers on the "Blood-corpuscle" throughout the Animal Series in the *Philosophical Transactions* for 1846. Here, I describe two forms of colourless corpuscles, which I designate respectively, the granule cell in the coarsely granular stage and the granule cell in the finely granular stage. Both kinds are seen to undergo change of shape after the blood is drawn from the body, but the latter in a more remarkable degree than the former. Thus, in the blood of the frog, I remark at page 67 that the granule cell, coarsely granular stage, exhibits change of shape with movements of the contained granules similar to what we see in the corresponding cell of the skate's blood, though not in so pronounced a degree; but the granule cell, finely granular stage, rapidly undergoes a more marked change of form by collapse and shooting out finger-like processes. This looks at first like a bursting of the cell and evacuation of its contents, but that it is no such thing can be easily proved by insinuating a minute quantity of water under the covering scale of glass, whereupon the collapsed, though apparently burst, cell becomes distended and is seen to be still entire.

All this is well known, but for a correct conception of the intimate nature of coagulation, the form-changes which the histological elements of the blood undergo in the process, require to be studied microscopically. The examinations for this purpose are best made on newly drawn human blood; but preliminary to this, we must make ourselves acquainted with the microscopical characters of the histological elements as they appear in blood flowing within the vessels of a living animal.

For such observations, the web of the bat's wing or frog's hind foot is the part which admits of being most conveniently displayed under the microscope, so that the correlations of the stream of blood in the arteries, capillaries, and veins may be watched for a length of time and truly traced without the interference of such disturbance of the natural course of the blood as is necessarily occasioned when an internal organ—the mesentery,[1] for example—is drawn out and made the object of examination.

Here I beg to refer to Plate IV. in my Essay on the State of the Blood and the Bloodvessels in Inflammation in *Guy's Hospital Reports* for October, 1850, in which the appearance of the blood stream is represented in an artery, in capillaries, and in a vein of the frog's web. I beg to refer also to the Plate illustrating my paper on the Rhythmical Contractility of the Veins of the Bat's Wing in the *Philosophical Transactions* for 1852, in which the appearance of the blood stream is represented in arteries and veins.

The stream of blood in the artery appears pale red, and homogeneous in the axis where the red corpuscles keep together, colourless next the walls of the vessel, when there is nothing but plasma and a few colourless corpuscles, perhaps, in it, rolling or sliding sluggishly along.

In the capillaries, in which the flow of blood is slower, the red corpuscles are seen to pass along in single file overlapping each other. Colourless corpuscles occur here and there interrupting the series of red corpuscles or, without interrupting it, press it aside, as may be seen in both bat and frog.

The stream of blood in the veins being comparatively slower than in the artery, not only the colorless but also the red corpuscles may be individually distinguished; the former in greater or less number next the walls of the vein, the latter in the axis of the stream.

In the bat, when the flow of blood is much retarded, the red corpuscles may be seen to aggregate in rouleaux, both in capillaries and in venous radicles. Of this I have drawings made from the life before me.

In viewing the flow of blood in the venous trunks of the bat's wing, it is an interesting spectacle to watch the backward eddy of the blood corpuscles in the sinuses of the valves, when, during the systole of the rhythmically contractile walls of those vessels, the valves are thrown into action to prevent regurgitation.

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[1] The mesentery of the frog, it is to be remembered, contains no capillaries. Small branches, recurrent from the arteries proceeding to the intestine, open directly into veins returning therefrom, as I originally pointed out.

A colourless corpuscle may sometimes be seen adhering by a point to the wall of a vessel, and to be by the force of the stream, bearing on it, pressed into a pear-shape, so that it presents the appearance of a minute polypus attached by a peduncle. When sticking by a greater extent of surface, the corpuscle is seen pressed into the shape of a rain-drop on the window. (See the wood-cuts at page 14 of my Essay on the State of the Blood and the Bloodvessels in Inflammation, before cited.) The adhesion to the wall of the vessel giving way, as it is seen to do, the colourless corpuscle, thus set free, immediately resumes its globular form, and is carried along in the stream.

It is particularly to be observed that the change of shape which a colourless corpuscle sticking by a point to the wall of the vessel may be seen thus to undergo by pressure on it from without, has nothing in common, and must not be confounded with the intrinsic changes of form which the colourless corpuscles in blood newly drawn from the body are seen to exhibit, as a moribund action, from an inherent endowment of their substance, under the influence of the altered conditions in which they are placed. *Within the living vessels, colourless corpuscles never undergo any such metamorphoses.* To this I have to add, that I never saw colourless corpuscles escape from the vessels by boring through their walls or in any other manner.

The more or less stellate form, which colourless corpuscles in blood out of the body (the granule cell, finely granular stage, especially) usually assume, would obviously not be well adapted for boring through the wall of a vessel or pushing its way in the interstices of a tissue. (Refer to my Papers on the Blood Corpuscle throughout the Animal Series, before cited.)

On the surface of the frog's mesentery, numerous white corpuscles belonging to the peritoneal fluid are found. Some of these under the stimulating influence of the exposure to the air, I have seen shooting out a single process, and executing slight locomotive changes besides.

Whenever and wherever, in a much inflamed part of the web of a frog, I have seen corpuscles—white or red or both—free, the walls of the vessels in which they had been contained had already given way and disappeared, as described at page 51 of my Essay on the State of the Blood and the Bloodvessels in Inflammation, before referred to. In the cases in which there was no longer a distinct vascular coat to be seen, the boundary of the channel along which the blood continued to flow was composed of colourless corpuscles, unchanged in shape, accumulated like stones on the sides of a torrent which had subsided after an overflow.

Inflammatory congestion, in a spot, is seen to take place by the close aggregation of the red blood corpuscles, intermingled with colourless ones, blocking up the vessels, while the plasma, being thereby excluded, drains away in the stream. This aggregation of blood corpuscles, which may be seen in the bat (see my Paper in the *Medico-Chirurgical Transactions* for 1853, on the State of the Blood and the Bloodvessels in Inflammation of the Web of the Bat's Wing), as well as in the frog, ends in stagnation of the current. Notwithstanding this, the force of the blood from behind does not appear to bear heavily on the stagnant mass, but the stream glides gently off by the collateral branches of the artery above. The only indication of pressure is the oscillation of the blood in the vessel below the place, where the stream passes off, as in the case of a divided artery.

In the case of a divided artery, it is to be observed that it is at the further margin of the wound where the congestion is greatest. This is owing to regurgitation in the vessels to which the collateral branches of the divided artery lead; direct *vis a tergo* bearing but little on the blood in the vessels in the nearer side of the wound. It was long ago pointed out by Dr. Jones in regard to the current in large arteries, to which a ligature had been applied, that "the blood does not continue to be impelled against the extremity of the artery with the same impetuosity with which it flowed through the vessel before it was tied. The blood is immediately determined into the collateral branches above."

Apparently, under the erroneous impression that as much *vis a tergo* as before ought to bear on the blood stagnant in the vessels of an inflamed spot by the stream of blood in the artery or arteries leading to it, the same distinguished authorities who tell us that colourless corpuscles bore through the wall of vessels, allege as a *fact* that the red corpuscles may be seen to be squeezed out through the walls of the gorged vessels.

The reality, of what seems to be here referred to, is described in my Essay on the State of the Blood and the Bloodvessels in *Guy's Hospital Reports* for October, 1850, before cited, at page 49, under the head of Resolution of Inflammation, as follows: The agglomerated and stagnant mass of blood-corpuscles in the extreme arterial branches, the capillaries, and venous radicles, exposed to the force from behind in the artery leading to those vessels, is seen to oscillate, yielding a little at each stroke of the heart, and again recoiling. The agglomerated mass being at last pushed onwards a little, some of it protrudes into the free vessels (into which the congested ones have an outlet), the stream in which detaches and carries away the corpuscles. In this manner successive portions of the agglomerated mass are carried away until, at last, being reduced to a small bulk, it is all driven onward by the stream from behind into the stream in front, where it is broken up into its constituent corpuscles which are carried away.

This process of resolution of inflammatory congestion is accelerated when the artery leading to the affected spot becomes dilated so that more *vis a tergo* is permitted to bear on the stagnant blood. A demonstration of this phenomenon can be easily made, as I state in my essay so often cited, by applying some irritating drop, such as a weak solution of sulphate of copper with a little vinum opii, to a congested web of a frog, whereupon the artery, after being constricted a little, soon becomes dilated and the process of resolution proceeds. This fact I have over and over again adduced as an interesting illustration of the *modus operandi* of stimulating collyria applied to the conjunctiva for the cure of pustular and catarrhal ophthalmia.[1]

Having thus described the blood as it is seen within the small vessels of the living bat and frog, we have next to examine it when newly drawn. For this purpose we can make use of human blood; and here I may observe that as human blood is similar to bat's blood out of the body, except that the corpuscles of the latter are smaller, there can be little doubt that the blood within the vessels of the human body comports itself in all essential respects in a manner similar to what we have seen in the case of bat's blood.

I have before me three drawings of a small drop of human blood, as viewed under the microscope with an eighth of an inch objective and second eyepiece, which I made with special care. The blood, being placed on a slide, was covered with a thin scale of glass and forthwith observed. In the first figure the blood is represented as it appeared on being immediately examined. The corpuscles, both the numerous red and the few colourless, dispersed confusedly about in the plasma whilst the colourless were already beginning to collapse and shoot out processes. In the second figure the same portion of blood is represented as seen when examined in about a minute or two after being first displayed under the microscope. The red corpuscles have became aggregated together like coins in rouleaux; the colourless corpuscles scattered about free from any adhesion to the red corpuscles, and though more changed in form still entire—none of them burst. In the interspaces between the rouleaux of the red corpuscles, there were perceived on careful adjustment of the microscope fine, pale, colourless fibres, shooting in every direction close on the surface of the glass slide.

The transition from the state of matters represented in the first figure to that represented in the second takes place as follows: the red corpuscles first overlap each other in linear series, then rising up on edge become fully applied to each other, face to face. The rolls of red corpuscles thus formed are disposed section-wise and represent an irregular network. The colourless corpuscles remain dispersed here and there in the meshes of this network.

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[1] I have also over and over again indicated constriction of small arteries as the first step to the establishment of congestion in the capillaries and venous radicles to which they lead, but ignorance of the correlations of the flow of blood in the extreme arteries, capillaries, and venous radicles, has stepped in to prevent a comprehension of the process.

The fine, pale, colourless fibres which make their appearance on the glass slide soon after the aggregation of the red corpuscles into rouleaux are the fibres of fibrine which has been deposited from the plasma. The fluid that now remains in the interspaces between the rouleaux of red corpuscles is, therefore, serum only.

It is thus not until the red corpuscles have aggregated into rolls that the deposition of fibrin takes place.

The colourless corpuscles, though collapsed, have not burst, and therefore have not given out their contents. They were still the same in number after the fibrin had been deposited as before. The rapidity and closeness with which the aggregation of red corpuscles into rolls takes place is much promoted, as we shall see, by the quantity in the plasma of the albuminous materials which by their combination form the solid fibrin. After the fibrin has been deposited from the plasma, therefore, and nothing but serum remains, the red corpuscles cease to adhere tenaciously to each other so that the rolls into which they had aggregated tend to become broken up. This may be demonstrated by gently touching the superjacent scale of glass with the point of a needle. In consequence of the agitation thereby occasioned, currents of serum and loose corpuscles take place, but it is at the same time observed that many of the red corpuscles being glued by a single point to the glass slide through the medium of the deposited fibrin are, as represented in the third figure which I have before me, pressed into a pear shape by the force of the currents of serum and loose corpuscles bearing on them.

The arrangement of the rolls of red corpuscles in a network is exhibited *sectionwise* when the drop of blood is thinly spread out as above described. When, however, the blood in the form of a drop is examined under the microscope with a lower but good penetrating power, the rolls into which the red corpuscles become aggregated are observed to be disposed in every direction in the form of a *spongework*, in the interstices of which the plasma is contained. The fibrin next consolidating forms the feltwork, in the interstices of which are inclosed the rolls of red corpuscles now become more or less disrupted while the serum is gradually squeezed out.

Coagulation usually begins in three or four minutes after the blood is drawn, and is completed within ten minutes. Though, in a cupful of blood newly drawn by venesection, the red corpuscles by virtue of their greater specific gravity tend to subside, their complete subsidence is arrested by the deposit of the fibrin which entangles them in its meshes. Thus it is that the clot remains red at the surface as well as at the bottom.

In the blood drawn in certain states of the system—inflammatory states especially—a subsidence of red corpuscles takes place before coagulation commences, leaving the plasma collected at the top.[1] This is owing not to the plasma coagulating more slowly, but to the more rapid sinking of the red corpuscles. In such a case, plasma while still fluid may be skimmed off in some quantity with a spoon.

If a drop of fluid plasma thus obtained be transferred to a slide and thinly spread out by being covered with a scale of glass and examined under the microscope, colourless corpuscles in considerable number will be seen. The colourless corpuscles thus do not sink with the red ones, but remain suspended in the plasma. Here, again, we can clearly perceive that, however much they may change shape, they do not burst, and that the pale delicate fibres of fibrin we see by and by deposited on the glass slide (now in greater quantity than was seen in the examination of a drop of entire blood) are not derived from them either bodily or by any action of their alleged evacuated contents on the plasma as a ferment determining the combination of fibrinogen and fibrinoplastic to form the fibrin fibres.

Turning to the examination of the remainder of the plasma which was skimmed off and left to itself, we find it coagulated into a buff coloured or grayish jelly-like mass, from which serum, after a while, may be observed to separate as from ordinary coagulated blood. In this clot, we have a mass of fibrin unmixed with red corpuscles, but in which there are interspersed numerous colourless corpuscles. The fibres of this fibrin examined under the microscope, it is not difficult to identify with the pale delicate fibres which, as above shown, we observe making their appearance, by deposition on the glass slide from the fluid plasma.

The collection of plasma at the top in consequence of the rapid sinking of the red corpuscles in blood drawn in certain abnormal states of the system constitutes the condition on which the formation of the "buffy coat" depends; the buffy coat being simply the plasma with the colourless corpuscles which remained collected at the top, after the subsidence of the red corpuscles, now become coagulated.

The sinking of the red corpuscles in the plasma of blood drawn in acute inflammation used to be supposed owing to slower coagulation than natural; but the fact is that coagulation of such blood occurs often more rapidly. Moreover the subsidence of the red corpuscles in the plasma to a degree sufficient for the formation of a buffy coat is found to have already taken place long before the time when blood normally coagulates after abstraction. The explanation of the more rapid subsidence of the red corpuscles is this: The disposition of the red corpuscles to aggregate together in rouleaux is very much augmented in blood on which a buffy coat is about to form. So much so is this the case in respect to rapidity that with whatever haste the drop of blood can be placed for examination under the microscope, the red corpuscles are found to have already aggregated; whilst in respect to closeness of aggregation, it is very much greater than usual. The effect of this increased rapidity and closeness of aggregation is that, being quickly collected together in compact masses, the red corpuscles sink in the plasma long before coagulation takes place. Again, it has been found that the condition of the blood which determines this more rapid and close aggregation of the red corpuscles is a more viscid and plastic state of the plasma arising from an increase in it of the quantity of the albuminous substances which combine to form the fibrin. The number of red corpuscles is at the same time less than normal.[1]

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[1] This occurs normally in the blood of the horse, and affords a good opportunity of obtaining a drop of the plasma for microscopical examination, in order to find among the colourless corpuscles, examples of the reddish nucleated cell which I describe and delineate (in my Papers on the "Blood-corpuscle," above cited) in the blood of the horse and elephant. Here I ought to refer to Mr. Gulliver's beautiful experiments, showing that the sinking of the red corpuscles proceeds with accelerated rapidity in a sort of arithmetical ratio.

In some inflammatory states of the system the plastic condition of the blood is increased though not to so great a degree as to lead to complete subsidence of the red corpuscles before coagulation. In such a case, though there is no actual buffy coat, there is a large quantity of fibrin at the top of the coagulated mass which by shrinking causes excavation of the surface, a condition of the clot designated simple cupping.

In reference to the separation of the red corpuscles from the plasma, it is to be remarked that it does not appear to be exclusively a separation by mere subsidence of the former any more than the retention of the colourless corpuscles at the top depends on their mere swimming in the plasma. Whilst the red corpuscles have an attraction for each other, the colourless are attracted by the plasma. This we observed indicated in the blood, as before mentioned, while circulating in the vessels of the bat's wing or in those of the frog's web, by the tendency of the red corpuscles to keep together in the axis of the stream, whilst in the stratum of plasma adjacent to the walls of the vessels, there may often be seen accumulations of colourless corpuscles, but no red ones.

Within the vessels in the living animal—both bat and frog—the red corpuscles not only keep together but tend to aggregate into rouleaux when the blood flows slowly or becomes altogether stagnant.

In regard to the colourless corpuscles, they tend, as we have seen, to collect on the inside of the walls of the vessels, where the stream of blood is slow, but this depends partly on an adhesion between them and the walls of the vessels.

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[1] See my various observations on the formation of the "Buffy Coat" in the *British and Foreign Medical Review* for October, 1842, the *Edinburgh Medical and Surgical Journal* for October, 1843, and *Guy's Hospital Reports* for October, 1850.

Colourless corpuscles, as before said, never undergo any amœba-like change of form within the vessels, but no sooner is blood drawn from the living body and examined under the microscope than such a change is found to have already commenced. But this change of shape, let it be repeated, is unaccompanied by bursting of the cell. After coagulation of the fibrin has taken place and the colourless corpuscles are now surrounded by serum alone, they tend by endosmotic absorption of the thinner fluid into their interior to resume a round form.

To recur to the mode in which coagulation of the blood contributes to the stopping up of the mouths of divided vessels and thereby to the arrestment of hemorrhage: The blood coagulating as it escapes, the clot which is formed in and around the retracted and constricted mouth of the bleeding artery is retained more firmly in its place by the interlacement of the fibres of the fibrin with those of the surrounding cellular tissue.

The condition on which the stoppage of bleeding from an artery depends, namely, retraction and constriction of its mouth, the more speedy coagulation of the arterial blood, and the greater firmness of the clot which is formed, are commensurate with the force to be resisted. If in the case of veins those conditions operate in a much less degree, so also is the force of the current to be stemmed very much less.

On the state of the blood o' the body generally at the time of the infliction of a wound dividing vessels, whether there be a sufficiency or deficiency of fibrinous materials, the formation of an external clot of sufficient firmness to arrest the bleeding greatly depends.

When bleeding from an artery is arrested by the formation of an external clot or by the application of a ligature, the stream of blood passes off, as before mentioned, by the first considerable branch above. Between this branch and the mouth of the vessel which is stopped up with the external clot or secured by the ligature, the artery is filled with blood. This collection of blood which presented itself as a glomerulus of red corpuscles next the mouth of the vessels, but higher up, is seen to consist chiefly of plasma with colourless corpuscles suspended in it, Dr. Jones called the *internal clot.* Petit had long before, under the idea that it was as operative in the suppression of the hemorrhage as the external clot, called it *bouchon*, whilst the external clot itself he called *couvercle.* Dr. Jones, however, showed that the internal clot plays no part in the arrestment of the bleeding, its formation being dependent on that event.

What Dr. Jones called the middle clot is of a different nature from either the external or internal clot, being composed of *exuded lymph.* The walls of the artery at the place of division, or at the place of ligature, becoming inflamed, this lymph is exuded and penetrates between the external and internal clots, or where the bleeding has been arrested by ligature, between the latter and the internal clot.

It is by the middle or lymph clot alone that the divided or ligatured artery becomes permanently closed, as was demonstrated by Dr. Jones. In the case of microscopical arteries, we see in the web of the frog that there is no special middle clot exactly, but the lymph exuded into the wound constitutes the material for the closure of the mouth of the vessel, as well as for the healing of the wound of the web.

Eventually the part of the artery occupied by the internal clot, and through which blood no longer flows, degenerates into a cord of cellular tissue, like what takes place in the case of the *ductus arteriosus*, *ductus venosus*, and the *umbilical arteries*, when the stream of blood is diverted from them.

A slough of the web of a frog having become detached, the network of vessels at the margin of the living part comes to present all the characters of a capillary network. The larger vessels, which have been truncated by the separation of the slough, become obliterated as far up as their connection with some considerable branch above, which now comes to represent the continuation, as the case may be, of the artery or principal radicle of the vein.

In microscopical examinations of frogs in which there existed sloughing and ulceration from inflammation excited by previous injury, I have often seen suddenly appear, in an artery of the sound web, glomeruli of red corpuscles, followed by loose flakes of fibrin holding together colourless corpuscles. Such glomeruli and flakes would perhaps block up the vessel for an instant or two, and then be forced on by the stream from behind, but to be succeeded by others. This appearance—of which I have drawings in my note-book before me—resembled somewhat the appearance within the caudal vein of the eel, of successive drops of red blood separated by the drops of lymph propelled into that vessel by the lymph heart.[1]

The embola referred to seem to be so many internal clots formed in the manner before described in connection with local congestions and stagnations, now become free in the general circulation, and similar, I believe, in their nature to the embola which are liable to occur in the human body after operations. They may block up any artery; but becoming at length diminished in bulk by successive disintegrations, as I have seen in the frog, they may eventually cease to cause any further obstruction. When, however, they happen to accumulate in and block up the pulmonary artery, death may be occasioned, as Sir J. Fayrer has found to be often the case after operations in India.

Sometimes I have seen only a grayish granulous plug consisting of colourless corpuscles held together by a tenacious looking fibrinous substance. A similar plug may be produced *at will* in the bat as well as in the frog, by pressing pretty firmly with a blunt point over an artery or vein under microscopical observation.

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[1] See my Paper entitled "The Caudal Heart of the Eel, a Lymphatic Heart," in the Philosophical Transactions for 1868.

The separation of the red corpuscles from the plasma, and consequently the formation of the fibrinous clots found in the heart and vessels after death, depend on the state of the blood before death in a manner similar to that which obtains in the formation of the buffy coat. Thus, in the bodies of persons who had died of pneumonia, inflammation of serous membranes, etc., in which the quantity of fibrinous material in the blood is increased and the disposition of the red corpuscles to aggregate consequently greater, the clots formed in the heart and large vessels were found by Rokitansky numerous, compact, and free from red corpuscles. Again, in cases in which, though there was no inflammatory alteration of the blood before death, the red corpuscles have been found to occupy the lower part of the clot corresponding with the most depending part of the body as it lay after death, so that here the separation had taken place by mere subsidence, in consequence of the greater slowness with which the blood coagulated within the vessels.

ARTICLE VIII.

GASTRO-HYSTERECTOMY, OR THE RECENT MODIFICATION OF THE CÆSAREAN SECTION BY DR. PORRO.

By ISAAC E. TAYLOR, M.D., Emeritus Professor of Obstetrics, and of the Diseases of Women and Children, Bellevue Hospital Medical College, New York.

I SELECT the word Hysterectomy as it expresses the true meaning of the act, the ablation or the removal of the uterus and ovaries, after the Cæsarean section is performed. Hysterotomy simply implies an incision without removal.

The medical profession are under many obligations to Dr. R. P. Harris, of Philadelphia, for his very valuable papers on the Cæsarean section, especially the last two, published in the *Americal Journal of the Medical Sciences*, for January and April, 1880. No writer has given more time, unwearied investigation, and closer attention to this important subject, and his information has been derived from various sources, not only in America and the British Isles, but also Continental Europe.

From his last two papers we glean that it is an absolute necessity, if the operation is to be successful, it must be attempted or resorted to as early as possible, or as early as the nature and circumstances of the case will admit, not only for the benefit and welfare of the mother, but of the child. Dr. Thomas Radford was a strong advocate for an early performance of the operation. It is however to Dr. Harris's untiring zeal, and to his able